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HYPERTENSION, LIPIDS AND PREVENTION

RHO-KINASE ACTIVITY IS INCREASED IN HYPERTENSIVE PATIENTS WITH LEFT VENTRICULAR HYPERTROPHY.

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Background: Rho kinase (ROCK) activation produces smooth muscle cell contraction, vasoconstriction and cardiovascular remodeling. Its role in human hypertension and left ventricular remodeling has not been evaluated. The aim was to compare circulating levels of ROCK in hypertensive patients with or without left ventricular hypertrophy (LVH).

Methods: Untreated essential hypertension patients (HT) with or without LVH were compared with normotensive healthy individuals. In all subjects were determined: ROCK activation in circulating leukocytes by measuring the levels of phosphorylated/total myosin light chain phosphatase 1 (MYPT1) by western blot and carotid to femoral pulse wave velocity (PWV). Left ventricular mass index (LVMI), relative wall thickness (RWT), left atrial (LA) size and mitral annulus tissue doppler were also measured by transthoracic echocardiography (LVH was defined as a LVMI > 115 grs/m2 in men and 95 > grs/m2 in women)

Results: mean ± SEM

	Controls (n = 34)	HT without LVH (n = 18)	HT with HVI (n = 14)
Age (years)	52 ± 1.1	51 ± 0.9	51 ± 0.9
Women (%)	52.9	50.1	56.5
BMI (Kg/m2)	25.1 ± 0.6	25.4 ± 0.6	26.5 ± 0.6
MAP (mmHg)	87 ± 1.2	120 ± 0.9*	121 ± 0.9*
MYPT1(p/t)	2.1 ± 0.4	2.8 ± 0.6	5.6 ± 0.9 *
PWV (m/seg)	8.1 ± 0.1	11.2 ± 0.4 *	11.3 ± 0.4 *
Septum (mm)	8.6 ± 0.3	9.1 ± 0.4	10.9 ± 0.4 *
LA (cm2)	17.6 ± 0.7	18.3 ± 0.8	21.2 ± 0.9 *
e/e´	8.6 ± 0.3	9.2 ± 0.5	11.2 ± 0.6 *
LVMI (gr/m2)	78 ± 2	82 ± 3	119 ± 5 *

* $p < 0.01$ vs controls after significant ANOVA. Abbreviations: MAP = mean arterial pressure, BMI = body mass index, p/t = phosphorylated / total.

HT patients with eccentric LVH (RWT < 0.42) showed increased Rho kinase activity vs no-eccentric LVH patients (7.8 ± 0.9 vs 2.9 ± 0.8, p= 0.003).

Conclusion: ROCK activation is evident in HT patients with echocardiographic evidence of LVH and pathological ventricular remodeling. The role of ROCK activation on the development and regression of LHV and cardiac remodeling should be further investigated.